activities of daily living, with the last patient being discharged on the fifth post-operative day. All have reported a marked symptomatic improvement.

The complex pathoanatomy of the mitral valve will present a difficult challenge for TMVR compared with TAVR. All the devices with early clinical experience have their advantages and disadvantages, and much work remains to be done before TMVR becomes a viable therapeutic alternative to surgical valve repair/replacement. However, the FIH experience with this system is a promising beginning. Stabilization by means of an apical tether is a novel way to attempt to minimize the risk of device migration, left ventricular outflow tract obstruction, and paravalvular leakage. The unique design features of this device also offer the potential for it to be useful across a wide range of mitral valve pathologies.

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Please note: Mr. Moat is a consultant for Medtronic and Tendyne Holdings, Inc. Drs. Blanke and Leipsic receive CT Core Lab service income through the University of British Columbia for Tendyne Holdings, Inc. Dr. Grayburn has received research grants from Abbott Vascular, Medtronic, Edwards Life-sciences, and Aastrom, and has Echo Core Lab service income through Baylor Research Institute for Tendyne Holdings, ValTech Cardio, and Guided Delivery Systems. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose. The authors thank Georg Lutter, Lucian Lozonschi, and Eduardo de Mar- chena as well as Dan Mans, Bob Vliulnd, Jeff Franco, and Jessica Kleine (and their colleagues at Tendyne) for contributions made to the development of the Tendyne system. Without them, this work would not have been possible.

**REFERENCES**


**Cardiovascular Screening in College Athletes**

Sudden cardiac death (SCD) is the leading cause of death in college athletes during sports (1). Pre-participation examinations are required in college athletes, although the best screening protocol to identify cardiovascular diseases known to cause SCD is debated. The purpose of this study was to examine results from a single National Collegiate Athletic Association (NCAA) Division I institution with extensive experience conducting electrocardiograms (ECGs) in the cardiovascular screening of athletes.

Routine 12-lead ECG screening of all intercollegiate athletes undergoing pre-participation evaluations at the University of Washington began in August 2010. The screening evaluation included a standardized history questionnaire from the Pre-Participation Physical Evaluation Monograph (4th edition), physical examination, and resting ECG. ECG interpretation was guided by modern standards, and screening abnormalities underwent additional evaluation in consultation with cardiovascular specialists.

Findings were compared among student athletes using the Fisher exact test, and statistical significance was defined as p < 0.05. The primary outcome measure was the identification of disorders associated

<table>
<thead>
<tr>
<th>TABLE 1 Patient Profiles</th>
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<tbody>
<tr>
<td>Patient #1</td>
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<tr>
<td>Age, yrs</td>
</tr>
<tr>
<td>Sex</td>
</tr>
<tr>
<td>Pre-TMVR NYHA functional class</td>
</tr>
<tr>
<td>Glomerular filtration rate, ml/min</td>
</tr>
<tr>
<td>Previous CABG</td>
</tr>
<tr>
<td>Heart failure hospital admission in previous 6 months</td>
</tr>
<tr>
<td>Etiology MR</td>
</tr>
<tr>
<td>Pre-TMVR MR grade (EROA, cm²)</td>
</tr>
<tr>
<td>Post-TMVR MR grade</td>
</tr>
<tr>
<td>Post-TMVR peak/mean mitral gradient, mm Hg</td>
</tr>
<tr>
<td>Length of hospital stay, days</td>
</tr>
<tr>
<td>Discharge destination</td>
</tr>
</tbody>
</table>

CABG = coronary artery bypass grafting; EROA = effective regurgitant orifice area; MR = mitral regurgitation; NYHA = New York Heart Association; TMVR = transcatheter mitral valve replacement.
with SCD. The Human Subjects Division at the University of Washington approved the study.

Data from 790 consecutive athletes screened between August 2010 and June 2014 were analyzed. Of the athletes, 56.2% were male, the mean age was 18 years (range, 17 to 25 years), 59.4% were Caucasian, 14.6% were African-American, 3% were Asian, 23.2% were of other/mixed race, and they participated in 19 different intercollegiate sports plus cheerleading.

At least 1 positive cardiovascular symptom or family history response was reported by 294 athletes (37.2%). Female athletes were more likely to report at least 1 positive symptom or family history response (43.3%) versus male athletes (32.4%) (p = 0.002). The most common history responses were syncope/symptomatic event (5.1%), and a family history of a heart problem, a heart murmur and 2 athletes (0.3%) with physical stigmata of Marfan syndrome.

Physical examination findings were abnormal in 28 athletes (3.5%), including 26 athletes (3.3%) with a heart murmur and 2 athletes (0.3%) with physical stigmata of Marfan syndrome.

ECG abnormalities were present in 22 athletes (2.8%). Male and female athletes (3.6% vs. 1.7%; p = 0.131), and African-American and Caucasian athletes (3.5% vs. 3.2%; p = 0.776) had similar rates of ECG abnormalities. The average time loss from sport to conduct secondary testing was 6.4 days (range, 0-33 days), and there were no adverse medical events from secondary testing or therapeutic procedures.

Five athletes (0.6%) were identified with cardiac conditions associated with SCD, including hypertrophic cardiomyopathy (n = 1), genetically confirmed long QT type I (n = 1), and Wolff-Parkinson-White syndrome (n = 3). All athletes with potentially lethal disorders were asymptomatic and had abnormal ECG findings (Table 1).

This analysis demonstrates that screening by history and physical examination alone has a low sensitivity to detect conditions associated with SCD in college athletes and that the addition of an ECG, when properly interpreted and with skilled cardiology resources, improves the detection of silent/congenital cardiac conditions associated with SCD. All 5 athletes in this cohort identified with a disorder

<table>
<thead>
<tr>
<th>Athlete</th>
<th>Diagnosis</th>
<th>Screening</th>
<th>Secondary Testing</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>19-year-old African-American male; basketball</td>
<td>Hypertrophic cardiomyopathy</td>
<td>History (−) Physical (−) ECG (−) Inferolateral ST-segment depression and deep T-wave inversion</td>
<td>Echo: IVS, 1.2 cm; posterior wall, 1.2 cm; LVEDD, 5.3 cm Cardiac MRI: apical wall thickness, 2.0 cm, with late gadolinium enhancement</td>
<td>Disqualified from competitive sports; underwent risk stratification, activity recommendations, and close follow-up</td>
</tr>
<tr>
<td>20-year-old Caucasian male; crew</td>
<td>Long QT syndrome</td>
<td>History (−) Physical (−) ECG (−) QTc interval, 465 ms</td>
<td>ETT: prolongation of the QTc interval at higher heart rates Genetic testing: confirmed KCNQ1 gene mutation for long QT type I</td>
<td>Extensive patient and family counseling by outside expert, declined beta-blocker therapy, allowed to continue rowing, no events over 4 yrs</td>
</tr>
<tr>
<td>18-year-old Caucasian male; baseball</td>
<td>Wolff-Parkinson-White</td>
<td>History (−) Physical (−) ECG (−) Ventricular preexcitation, short PR interval, delta wave</td>
<td>Echo: normal EP study; high-risk accessory pathway; status post-ablation</td>
<td>Returned to sport</td>
</tr>
<tr>
<td>18-year-old Caucasian male; baseball</td>
<td>Wolff-Parkinson-White</td>
<td>History (−) Physical (−) ECG (−) Ventricular preexcitation, short PR interval, delta wave</td>
<td>ETT: abrupt loss of pre-excitation (low-risk pathway)</td>
<td>Returned to sport</td>
</tr>
<tr>
<td>18-year-old Caucasian female; basketball</td>
<td>Wolff-Parkinson-White</td>
<td>History (−) Physical (−) Murmur ECG (−) Ventricular preexcitation, short PR interval, delta wave</td>
<td>Echo: normal ETT: abrupt loss of pre-excitation</td>
<td>Returned to sport, interval episode of palpitations and near-syncope led to EP study with ablation, returned to sport</td>
</tr>
</tbody>
</table>

(−) = negative; (+) = positive; ECG = electrocardiogram; Echo = echocardiogram; EP = electrophysiological; ETT = exercise treadmill test; IVS = interventricular septum; LVEDD = left ventricular end-diastolic diameter; MRI = magnetic resonance imaging.
associated with SCD were detected by an ECG and would have been missed by only a screening history and physical examination.

This analysis exhibits the rather vague nature and low yield of screening questionnaires. More than one-third of athletes reported at least 1 positive cardiac symptom or family history response. The American Heart Association recently expanded their primary recommendations for screening from a 12-point to a 14-point assessment (2). However, simply asking more questions is unlikely to improve detection of athletes at risk when the sensitivity and specificity of the tool itself have considerable limitations.

SCD in NCAA athletes is more frequent than initial estimates, with an overall incidence of 1:43,000, with male basketball players having the highest risk of SCD at 1:7,000 athletes per year (1). Strong consideration must be given to implementing improved models of prevention.

Accurate interpretation of an athlete’s ECG requires proper training and experience. The false-positive rate in this study was only 2.2%, and approximately 1 in 4 athletes with abnormal ECG findings were found to have a cardiac disorder associated with SCD. Physician expertise, cardiology, and institutional resources vary among NCAA institutions, which will affect both the capacity and ability to implement ECG screening. Thus, the findings of this study may not be applicable to institutions with less experience. If an ECG is included in the cardiovascular screening of athletes, it must be interpreted with modern standards that distinguish physiological cardiac remodeling from findings suggestive of underlying cardiac pathology and be conducted with adequate cardiology oversight and resources to assist with the secondary investigation of ECG abnormalities.

Screening history questionnaires have a high response rate, and their value in the detection of athletes at risk when used as the sole screening tool is uncertain. ECG screening increases the ability to identify athletes with disorders associated with SCD and thus meet the primary objective of pre-participation screening. An integrated cardiovascular screening that includes an ECG should be considered best practice for the pre-participation evaluation of college athletes.

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http://dx.doi.org/10.1016/j.jacc.2015.02.072

Please note: The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

REFERENCES


Myocardial Edema Should Be Stratified According to the State of Cardiomyocytes Within the Ischemic Region

We read the article by Fernández-Jiménez et al. (1) published in the Journal with great interest. The investigators conclude that the evolution of myocardial edema within a week after ischemia/reperfusion (I/R) follows a bimodal pattern: T2 relaxation times and water content reach the highest level at 2 h of reperfusion in the ischemia area, decrease to the lowest at 24 h, and rise to a peak again on day 7. Myocardial edema, namely a bright signal intensity zone on T2-weighted short tau inversion recovery cardiac magnetic resonance, is referred to as an area at risk that consists of irreversible and reversible injured tissue. The cardiomyocytes located in the irreversible region are necrotic and unsalvageable. Irreversible region is surrounded by a reversible region encompassing cells that are integrated without membrane disruption and can recover structure and function after reperfusion. A severe degree of edema will overwhelm the necrotic cells when reperfusion is